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(54) Apparatus for reversion of tachyarrhythmia including post therapy pacing delay

Vorrichtung zur Beendigung von Tachyarrythmien einschliesslich Nachtherapie-Stimulierungsverzögerung

Appareil pour la suppression de tachyarythmie incluant un délai de stimulation postthérapeutique

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Description

Technical Field

This invention relates to implantable medical devices which monitor the cardiac state of a patient by sensing sinus rhythm, ventricular tachycardia and ventricular fibrillation and which deliver therapy in the form of electrical energy to cardiac tissue to revert tachycardia and restore sinus rhythm.

As used herein antitachycardia pacing refers to any pacing for the reversion of tachycardia. The term tachyarrhythmia refers to any fast abnormal rhythm of the heart which may be amenable to treatment by electrical discharges. This specifically includes ventricular tachycardia (VT), supraventricular tachycardia (SVT), ventricular flutter, ventricular fibrillation (VF), atrial tachycardia (AT), atrial flutter and atrial fibrillation (AF).

The term therapy as used herein includes the processes used between the detection and the reversion of a tachyarrhythmia and includes the actions of antitachycardia pacing, cardioversion and/or defibrillation shocks. The term cardioversion refers to the discharge of electrical energy into the cardiac tissue in an attempt to terminate or revert a tachyarrhythmia. This may take the form of a high energy discharge (up to 40 Joules or more) or a low energy discharge (less than 1 Joule). Cardioversion shocks may or may not be synchronized to the rhythm of the heart. Defibrillation is a particular example of cardioversion.

This invention applies equally to devices which deliver energy synchronized to an R-wave and to those that do not, and it applies to devices which use lower energy pulses as well as to devices which use higher energy pulses. The invention applies to devices which deliver cardioverting shocks alone as well as to devices which deliver antitachycardia pacing pulses alone or in combination with cardioverting shocks. The invention will usually apply to implantable ventricular cardioverters, but is equally applicable to atrial cardioverters or multiple chamber cardioverters or defibrillators. The invention applies also to the delivery of any antitachycardia pacing pulses and post reversion pacing therapy.

Background Art

An example of an implantable cardioverting device is in Patent No. 3,952,750 to Mirowski et al.

This device uses an AGC (Automatic Gain Control) for sensitivity adjustment. An analog signal is used as a basis for adjustment. Signal averaging is performed on peak amplitudes and the sensitivity threshold is automatically adjusted by this process. However, this device does not perform any bradycardia support and was designed to detect VF (ventricular fibrillation) only. That is, it was not designed for detection of both VF and sinus rhythm and does not operate effectively as a detection device for bradycardia support and normal sinus rhythm.

A problem also exists in AGC devices where a VF has a variable amplitude. The presence of occasional peaks in its waveform in addition to lower "sub threshold" amplitudes is known to automatically adjust on the basis of the peaks with the effect of ignoring the lower amplitudes of the waveform, hence producing an erroneous result. Thus, an incorrect sensitivity adjustment is performed. VF is therefore undetected and could be mistaken for sinus rhythm. This may cause severe difficulties, discomfort and may even cause the death of the patient.

If all VF signals were low peak, the AGC would give a correct result, but it does not allow for the existence of occasional high peaks. Thus the device could erroneously operate on the basis that sinus rhythm had been restored. A further problem with AGC devices is that they fail to detect rapidly changing amplitudes which are often observed during VF.

Furthermore, AGC devices are not generally designed to cope with bradycardia support or the condition of asystole.

If there is no signal response and asystole is present, an AGC device could automatically increase detector sensitivity until unwanted noise signals are picked up, such as muscle noise, electrical noise, etc., and these could be recognized by the device as being an arrhythmia. The device would then cause the application of incorrect therapy, causing great discomfort and possibly death of a patient. This is a reason why the device has not generally been implanted in patients suffering from the effects of asystole and who need a device designed for bradycardia support.

As a result of these shortcomings, an AGC has not been effective in detection in implantable devices in patients who suffer from asystole and require bradycardia support, in addition to antitachyarrhythmia therapy. In addition to antitachyarrhythmia devices, there are some existing pacemaker devices which include an AGC for sensitivity adjustments. These pacemaker devices may be effective in organized heart rhythm detection, but are not designed to allow for VF detection or for differentiation between VF and asystole.

Another prior art detection device is described in United States Patent No. 4,184,493 to Langer et al. and relates to VF detection using the principle of a probability-density function. In this device, the ECG is filtered by a high pass filter after which the filtered ECG is used to derive the control voltage for an automatic gain control circuit. However, this device does not overcome the problem of detection of lower amplitude or fine VF's due to the high pass filtering of the VF signal. Furthermore, the device is not designed for bradycardia support of patients experiencing asystole.

An article in The American Journal of Cardiology Vol. 52, page 265, entitled "The Automatic Implantable Defibrillator: Local Ventricular Bipolar Sensing to Detect Ventricular Tachycardia and Fibrillation" by Winkle et al.

refers at page 270 to the use of rate detection circuits in conjunction with a morphology dependent criterion, such as the probability-density function to minimize the possibility of delivery of shocks during sinus or other narrow-QRS SVT. The article then states that the "addition of a morphology-dependent criterion is done at the expense of increasing the likelihood that some VT's will not be recognized by the system" and that the device "may occasionally deliver a shock for a a sinus or other SVT". This further emphasizes the need for a device which reliably detects VT and sinus rhythm and is also designed for bradycardia support pacing.

Another prior art heart rate detection device is shown in U.S. Patent No. 4,393,877 to Imran. This heart rate detection apparatus includes two mutually inclusive detector circuits responsive to ECG waveforms of different characteristics. One detection circuit is responsive to ECG waves with high slew rates, or spiky waves, while the other detection circuit is responsive to ECG waves with low slew rates, or sinusoidal waves. This device, however, is not designed to distinguish between low amplitude VF and asystole. Also the device is not designed to overcome the problem of double sensing after a defibrillation shock due to far-field R-waves or current of injury T-waves which may occur, for example when, post shock, the T-wave is, for a period of time, of a higher amplitude than the R-wave.

Furthermore, some attempts have been made at permanently increasing the sensitivity to overcome the problem of loss of low amplitude VF signals. This has resulted in the additional problem of double-sensing of VT and sinus rhythm signals, causing erroneous results in the detection device which can produce an incorrect therapy, causing great discomfort and possibly patient death.

Another problem with existing detection devices can be caused by the presence of a current of injury T-wave. It is known that after therapy (shock or pacing) when a tachyarrhythmia has been reverted and sinus rhythm has been restored, the T-wave has, for a time, an amplitude which may be as high as or higher than that of the R-wave, thereby causing a double counting effect. Hence there may be at times a need in antitachyarrhythmia devices to differentiate between R-waves and current of injury T-waves in initial post therapy sinus rhythm.

Even if such differentiation can be successfully accomplished, and results in the proper application of appropriate therapy, a problem still exists with respect to providing post therapy bradycardia support pacing so that further arrhythmias are not produced.

U.S. Patent No. 4,561,442 to Vollmann et al. entitled "Implantable Cardiac Pacer with continuous Microprocessor Programmable Antitachycardia Mechanisms and Patient Data Telemetry" describes a cardiac pacer which, on detection of a tachycardia, initiates a preprogrammed automatic antitachycardia mechanism. When the tachycardia is terminated, the pacer reverts to its normal pacing mode at the programmed minimum rate.

A tachycardia reversion pacer is also disclosed in U.S. Patent No. 4,587,970 to Holley et al. This device generates a sequence of pacing pulses at intervals which are determined by the effective refractory period and the presence or otherwise of an unevoked heartbeat during a preceding pulse burst. At termination of the tachycardia, pacing pulses continue to be generated at increasing pacing intervals until they merge into standby pacing, thereby providing a smooth transition between the fast pulses used to terminate the tachycardia episode and subsequent beating in sinus rhythm.

Supraventricular tachycardias have been observed after termination of ventricular tachycardia by both cardioversion and antitachycardia pacing as described in the article "Comparative Efficacy of Transvenous Cardioversion and Pacing in Patients With Sustained Ventricular Tachycardia: A Prospective, Randomized, Crossover Study" by Saksena et al. in Circulation, Vol. 72, No. 1, pp. 153-160 (1985). Termination with transvenous cardioversion was followed by occurrences of atrial fibrillation, atrial flutter, and sinus tachycardia while one incidence of sustained sinus tachycardia was observed after termination of ventricular tachycardia with rapid ventricular pacing.

Post cardioversion arrythmias have also been observed by Saksena and Calvo as referred to in the article "Transvenous Cardioversion and Defibrillation of Ventricular Tachyarrhythmias: Current Status and Future Directions" in PACE, Vol. 8, pp. 715-731 (1985). In the study, the incidence of supraventricular tachyarrhythmias was substantial and transient bradyarrhythmias were also frequent after transvenous cardioversion.

It is therefore apparent that the heart is susceptible to arrythmias after antitachycardia therapy in the case of both antitachycardia pacing therapy and cardioversion shock therapy.

In patients receiving both antitachycardia therapy and bradycardia support pacing, it has been observed in some cases that such post therapy arrhythmias have been attributable to the lack of a sufficient time delay between the antitachycardia therapy and the bradycardia support pacing.

Following successful antitachycardia pacing or cardioversion shock therapy, the heart's conduction system is disorganized for a period of time often longer than the pacing standby interval of, for example, 857ms (70bpm), and bradycardia support pacing given too soon after such therapy while the heart is still in a disorganized state can easily reinduce the arrhythmia or initiate a new arrhythmia.

It is an object of the invention to reduce the likelihood of reinducing or inducing an arrhythmia by allowing a time delay after successful antitachycardia therapy before bradycardia support pacing is restarted. The time period for the post period therapy delay must be substantially longer than the normal pacing standby

interval.

EP-A-0 324 604, which is prior art under Article 54(3) EPC, discloses an apparatus for detecting arrhythmia comprising tachyarrhythmia detection means and tachyarrhythmia confirmation means. Bradycardia support pacing is inhibited for programmable periods of time after antitachycardia pacing or defibrillation which reverts a tachyarrhythmia so as to avoid any pro-arrhythmic effect.

The present invention provides an apparatus for reversion of tachyarrhytimia comprising:

bradycardia support pacing means for providing bradycardia support pacing;
detection means for detecting the presence of a tachyarrhythmia;
therapy for delivering antitachyarrhythmia therapy;
confirmation means for providing confirmation of the reversion of said tachyrhythmia following delivery of said antitachyarrhythmia therapy;
delay means for delaying, during a programmed and un-interruptible delay period substantially longer than one normal bradycardia support pacing interval, any determination of a need for therapy and any provision of therapy after said confirmation means has provided said confirmation of the reversion of said tachyarrhythmia; and
means for automatically switching said apparatus into a bradycardia support pacing mode at the end of said delay period and for delivering any needed bradycardia support pacing using substantially a normal bradycardia support pacing standby interval, said automatic switching of said apparatus into said bradycardia support pacing mode occurring without further determination of whether or not there is a need for further antitachycardia therapy.

Preferred features of the invention are set out in dependent claims 2 to 10.

This delay period varies from one patient to the next and depends on the condition of the patient, the types of arrhythmia, the type of therapy given and other factors. The most successful delay periods have been found to be in the range of 2 to 5 seconds, although in some cases the delay period may fall outside this range. In all cases, the magnitude of the delay period is substantially greater than the bradycardia support pacing standby interval, which is usually of the order of 857ms (less than 1 second). It is preferable, therefore, that the delay period is a programmable parameter to allow for a physician to select a suitable value for each individual patient.

Further objects, features and advantages of the invention will become apparent upon consideration of the following detailed description in conjunction with the drawings, in which:

FIG. 1 is a block diagram of an arrhythmia control system in which the present invention may be used;
FIG. 2 is a block diagram of the pacemaker of FIG. 1;
FIG. 3 is a block diagram of the microprocessor of FIG. 1;
FIG. 4 is a logic flow diagram of the software executed by the microprocessor of FIG. 3 in accordance with the invention;
FIG. 5 is a logic flow chart of the low sensitivity subroutine of FIG. 4;
FIG. 6 is a logic flow diagram of the post therapy delay subroutine of FIG. 4;
FIG. 7 depicts an ECG trace outlining multiple sensitivity responses to various cardiac conditions and therapies;
FIG. 8 depicts another ECG trace outlining additional multiple sensitivity responses to various cardiac conditions and therapies;
FIG. 9A depicts an ECG trace outlining bradycardia pacing after defibrillation shock with post shock delay; and
FIG. 9B depicts an ECG trace outlining bradycardia pacing after antitachycardia pacing with post antitachycardia therapy pacing delay.

Best Mode for Carrying Out the Invention

Referring to FIG. 1, there is depicted a block diagram of an arrhythmia control system 10. System 10 is designed to be implantable and includes a pulse module 11 and appropriate leads. More particularly, system 10 will generally include a cardiac lead extending to the atrium of a patient's heart for the administration of therapy to the atrium or a cardiac lead 12 extending to the ventricle of a patient's heart 14 for the administration of therapy to the ventricle. System 10 generally also includes a pacemaker 15 for the detection of analog signals representing cardiac electrical activity and for the delivery of pacing pulses to the heart; a microprocessor 16 which, in response to various inputs received from the pacemaker 15 as well as from a defibrillator 17, performs various operations so as to generate different control and data outputs to both pacemaker 15 and defibrillator 17; and a power supply 18 for the provision of a reliable voltage level to pacemaker 15, microprocessor 16 and defibrillator 17 by suitable electrical conductors (not shown). Defibrillator 17 produces a high voltage to charge its capacitors and then discharges them in response to control signals from microprocessor 16. A defibrillator electrode lead 19 transfers the energy of a defibrillator shock 20 from the implanted pulse module to the surface of the heart 14.

Microprocessor 16 is connected to an external memory 21 by an address and data bus 22. An end-of-life (EOL) signal line 24 is used to provide, to microprocessor 16, a logic signal indicative of the approach of battery failure in power supply 18.

As more fully described below, microprocessor 16

and pacemaker 15 are connected by a communication bus 25, a sense line 26, a pace control line 27, a sensitivity control bus 28, and a pacing energy control 29. As also more fully described below, microprocessor 16 is connected to defibrillator 17 by a charge level line 30, a charge control bus 31, a shock control bus 32, and a dump control bus 34.

Referring to FIG. 2, pacemaker 15 comprises pacing circuit 35 which includes a pacing pulse generator 36, sensing circuit 37, and telemetry circuit 38. In addition, there is a control block 39 which includes an interface to microprocessor 16.

In operation, sensing circuit 37 detects analog signals 40 from the heart 14 in an internal QRS detector 37A and converts the detected signals to digital signals. Furthermore, sensing circuit 37 receives an input sense control signal (which determines the sensitivity of the detection circuits in sensing circuit 37) by way of a sense control bus 41 from control block 39. As more fully described below, a change in this sensitivity will affect the voltage deviation required at the sensing electrode for a sense to be registered.

Pacing circuit 35 also receives inputs from control block 39 including a pace control and a pacing energy control by way of pacing control bus 42 which carries the signals on pace control line 27 and pacing energy control bus 29. The pace control determines the type of pacing to occur while the magnitude of the pulse energy is determined by the pacing energy control. Pacing circuit 35 causes pulse generator 36 to generate the pacing pulse 44 which is delivered to the patient's heart 14 by means of cardiac lead 12.

Telemetry circuit 38 provides a bi-directional link between control block 39 of pacemaker 15 and an external device such as a programmer. It allows data such as the operating parameters to be read from or altered in the implanted pulse module 11.

Referring to FIG. 3, microprocessor 16 comprises two 16-bit timers 47 and 48, CPU 49, vectored interrupt block 50, RAM 54, ROM 55, ports interface 57 and an internal communications bus 58. RAM 54 acts as a scratch pad and active memory during execution of various programs stored in ROM 55 and used by microprocessor 16. These programs include system supervisory programs, detection algorithms for detecting various arrhythmias, and programming implementing the logic flow diagram of FIG. 4, as well as storage programs for storing, in external memory 21, data concerning the functioning of module 11 and the electrogram provided by cardiac lead 12. Timers 47 and 48 and associated control software implement some timing functions required by microprocessor 16 without resort entirely to software, thus reducing computational loads on and power dissipation by CPU 49.

Signals received from telemetry circuit 38 permit an external programmer (not shown) to change the operating parameters of pacemaker 15 by supplying appropriate signals to control block 39. Communications bus 25 serves to provide signals indicative of such control to microprocessor 16. Thus, it is also possible for an external programmer to control operation of defibrillator 17 by means of signals provided to microprocessor 16.

Appropriate telemetry commands may cause telemetry circuit 38 to transmit data to the external programmer. Data stored is read out, by microprocessor 16, on to communications bus 25, through control block 39 in pacemaker 15, and into control block 38 for transmission to the external programmer by a transmitter in telemetry circuit 38.

Microprocessor 16 receives various status and/or control inputs from pacemaker 15 and defibrillator 17. During normal pacer operations the input signal to pacemaker 15 is a sense signal on sense line 26 which is used by microprocessor 16 to perform operations such as arrhythmia detection. Microprocessor 16 produces outputs such as the pace control on pace control line 27 which determines the type of pacing to take place. Other pacemaker control outputs generated by microprocessor 16 include a pacing energy control signal on pacing energy control bus 29 which determines the magnitude of the pulse energy, and a sensitivity control signal on sensitivity control bus 28, which determines the sensitivity setting of the sensing circuit.

Microprocessor 16 provides to defibrillator 17 a shock control signal on shock control line 32 which indicates that a shock is to be delivered to the patient, a dump control signal on dump control line 34 which indicates that a shock is to be dumped at an internal load within defibrillator 17, and a charge control signal on charge control bus 31 which determines the voltage level of the shock to be delivered. Charge voltage level line 30 provides a digital signal representative of charge voltage from an analog to digital converter within defibrillator 17, thus providing a feedback loop which assures that a shock of proper energy level is delivered by defibrillator 17.

FIG. 4 is a logic diagram of the microprocessor flow control for controlling the sensitivity of sensing circuit 37, with the start being shown at 90. At 90A, the sensitivity is set to medium gain. At 91, a determination is made as to whether bradycardia is detected or not. If bradycardia is detected, as shown at 91A, then bradycardia pacing is delivered at 92. This cycle continues until bradycardia ceases to be detected, as shown at 91B. A determination is then made as to whether or not tachyarrhythmia has been detected at 93. If tachyarrhythmia is not detected, as shown at 93A, the program will loop back to 91, and there will be no change or delivering of therapy until the detection of either bradycardia or tachyarrhythmia. If tachyarrhythmia is detected, as shown at 93B, then the sensitivity setting is switched to the medium setting at 94 (if not already at that setting).

A decision with respect to tachyarrhythmia confirmation then takes place at 95. If a tachyarrhythmia has been confirmed, as shown at 95A, then antitachyarrhythmia therapy is delivered to the patient, at 96. This

antitachyarrhythmia therapy may take the form of defibrillation shock therapy or a train of antitachycardia pacing pulses.

The time limit for the application of antitachyarrhythmia pacing therapy at 96, prior to the delivery of a shock, is of importance. In this regard, reference is made to United Kingdom Patent Application Serial No. 8816578.2 of Richard Grevis and Loraine Holley, filed July 12, 1988 and entitled "Arrangement for Treating Tachyarrhythmias of the Heart", assigned to the assignee of the present invention. In this application, the time limit for application of a shock is determined in accordance with the haemodynamic condition of the patient.

The pacing operation at 96, and more specifically, the manner in which the pacing energy is changed in accordance with events which have occurred, is described in copending European Patent Application Serial No. 89300232.9 of Norma Louise Gilli, filed January 11, 1989, entitled "Apparatus and Method for Controlling Pulse Energy in Antitachyarrhythmia and Bradycardia Pacing Device," also assigned to the assignee of the present invention.

On completion of the antitachyarrhythmia therapy, the program executes a low gain subroutine 101, described below with respect to FIG. 5, and then loops back to 94, where sensitivity is set to medium. The program then passes back to 95 for the decision of tachyarrhythmia confirmation.

If there is no confirmation of the tachyarrhythmia at 95, as shown at 95B, then the decision is made at 97 as to whether the condition of asystole is present in the patient. If there is no asystole, as shown at 97A, then the loop passes back to 91 by way of a post therapy delay subroutine 102 described below with respect to FIG. 6, and the cycle starts again with the decision of bradycardia detection and the cycle which follows from there.

If at 97 there appears to be asystole as shown at 97B, it is of importance to realize that this may be due to the absence of a signal when the sensitivity is at the medium setting. The next step is to confirm whether the condition of asystole is, in fact, present. The reason for this is to differentiate between asystole and a fine VF which would not normally produce a signal in the medium sensitivity setting. To achieve differentiation, switching of the sensitivity to the high setting occurs at 98. Following the increase in the sensitivity level, a confirmation of tachyarrhythmia takes place at 99. It is at this time that a differentiation between asystole and a tachyarrhythmia occurs. If the condition is a tachyarrhythmia, as shown at 99A, then antitachyarrhythmia therapy is delivered at 96 in the form of defibrillation shock therapy or antitachycardia pacing. The program then loops back to 94 as previously described.

If there is no confirmation of a tachyarrhythmia at 99, as shown at 99B, then there is either the condition of asystole or the tachyarrhythmia has been reverted. At this time, the sensitivity is switched back to the medium setting at 100, and the loop passes back to 91, by way of subroutine 102, for the detection of bradycardia and the cycle as previously described. It is desirable that bradycardia support pacing be inhibited for a programmable period of time after reversion of a tachyarrhythmia to avoid any pro-arrythmic effect. Such delay may be implemented in one of timers 47 and 48 or in software, as in subroutine 102, as described below with respect to FIG. 6.

Referring to FIG. 5, in order to distinguish between sinus rhythm and a current of injury T-wave it is necessary to temporarily adjust the detector circuit to a low sensitivity level. This is accomplished by the low sensitivity subroutine IOI. After the subroutine is accessed and started, the gain is temporarily set low at 103. This low sensitivity setting is lower than the medium sensitivity setting, which in turn, is lower than the high sensitivity setting. Immediately after the gain is set to the low level a determination is made as to whether there is sinus rhythm at 104. If there is no sinus rhythm, the subroutine branches from 104A to RETURN and control is returned to the main program. However, if sinus rhythm is detected (104B), a determination is made at 105 as to whether a predetermined time interval since the delivery of tachyarrhythmia therapy has expired. This time interval may be programmed by the physician to account for the time required in the particular patient for the current of injury T-wave to be diminished to a level at which the detector will not trigger as if it is a QRS complex when the sensitivity is set to the medium level. If this time has elapsed (105B), the subroutine is terminated and control is returned to the main program. However, if this time has not elapsed, the subroutine loops from 105A back to 103 where the gain is again set at the low level. As long as sinus rhythm is detected and timeout has not occurred, the gain remains low. However, after the timeout occurs the subroutine is terminated and control is transferred back to the program of FIG. 4.

Referring to FIG. 6, the post therapy delay subroutine 102 is described. Since subroutine 102 is located in the return loop to step 91 for both reversion of a tachyarrhythmia at 97A and for lack of confirmation of a tachyarrhythmia at 99B (FIG. 4), it is necessary that there exist a mode wherein no delay is introduced. Specifically, if a tachyarrhythmia is detected at 93 but there is no confirmation at 95 and apparent asystole at 97 and no confirmation at 99 (even though the sensitivity has been set to the highest level at 98) then a tachyarrhythmia has led directly to asystole. In this case, since no therapy was administered, a delay in pacing is undesirable. Therefore when tachyarrhythmia therapy of any kind is delivered, a flag (not shown in Fig. 4) is set. This flag is read at step 106 to determine whether therapy has been delivered. If the answer is NO (106A) then control is immediately returned to the program of FIG. 4 and no delay is introduced. However, if the flag indicates that therapy has been administered, branching to 106B

occurs. The flag is reset at 107. At 108 a determination is made as to whether a shock or pacing was last delivered. If the answer to the inquiry at 108 is YES then the program branches at 108A to a post shock delay 109A. After this delay, control is returned to the program of FIG. 4. The length of the post shock delay at 109A is programmable and is typically set so that the time between termination of tachyarrhythmia therapy at 96 and the delivery of bradycardia pacing at 92 is approximately 4.0 seconds. The actual delay introduced at 109A may be just over 2 seconds with the remainder of the delay being generated by low gain subroutine 102 which requires some time to determine whether sinus rhythm is present at 104 (FIG. 5) and bradycardia detection at 91 which typically has a programmed escape or standby interval of approximately 857ms.

If a shock has not been delivered at 108 then antitachycardia pacing has occurred. Subroutine 102 proceeds from 108B to 109B where a post pacing delay is introduced. The length of the post pacing delay is programmable but is generally selected so that the total delay from delivery of tachyarrhythmia pacing at 96 to the delivery of bradycardia pacing at 92 is in the order of 3.0 seconds. Specifically, the post pacing delay introduced at 109B is typically in the order of 1.0 seconds. As is the case for the delay introduced at 109A, the remainder of the delay is made up by the time required to recognize the absence of sinus rhythm at 104 and the escape or standby interval of bradycardia detection at 91.

It will be understood that if sinus rhythm is detected at 104 (Fig. 5) the additional delay of approximately one or two seconds introduced at 109A or 109B will be of little consequence. In this case, if a normal sinus rhythm has been restored, bradycardia will not be detected at 91 and pacing is not delivered in any event. However, should a bradycardia condition commence after the establishment of normal sinus rhythm by the delivery of tachyarrhythmia therapy, the additional delay of one or two seconds will not be critical. If it is desirable to avoid even this delay, the flag set when tachyarrhythmia therapy is delivered at 96 can be reset if normal sinus rhythm is detected at 104.

Referring to FIG. 7, the ECG trace shows a VF detected at 110, the sensitivity is set at medium and the TDO (tachycardia detection output provided by the algorithms used in microprocessor 16) is high showing a positive response from the medium sensitivity signal.

At 111, there is a reconfirmation of the VF. The reconfirmation is positive (the TDO still shows a high reading) and therefore the sensitivity setting is sufficient and it remains at the medium level.

At 112, a defibrillation shock is given. Immediately post shock 112, low sensitivity subroutine IOI is accessed and the TDO shows a low reading. The sensitivity is then switched from low sensitivity to medium sensitivity (FIG. 4, at 98) and then from medium sensitivity to high sensitivity (FIG. 4, at 94). The high sensitivity then shows a high TDO reading indicating detection of the presence of a fine (low amplitude) VF which was not detected at the low or medium sensitivity levels.

Reconfirmation is given at 113 (FIG. 4, at 99) showing the continued presence of the fine VF, as there is still a high reading on the TDO. Therefore at this stage the high sensitivity level remains in force.

At 114, defibrillation shock therapy is given. Immediately post shock 114, the low sensitivity subroutine IOIB is again executed. Since sinus rhythm is not detected, the sensitivity returns to the medium level (FIG. 4, at 94) and then to the high level (FIG. 4, at 98) to pick up any fine VF which may be present. Post shock, the low TDO signal shows the absence or reversion of the VF and the presence of asystole, rather than VF as no waveform is detected at the high sensitivity level. Since the TDO is low at the high sensitivity level, asystole is therefore assumed to be present. VVI pacing treatment is then given at 115.

At 116, sinus rhythm is detected as a result of the pacing therapy, with the sensitivity switched to the medium sensitivity level. It is not necessary to use low sensitivity to distinguish between R-waves and current of injury T-waves in this late occuring sinus rhythm complex.

Referring to FIG. 8, a VF is detected at 120, the sensitivity signal is set at medium sensitivity and a high TDO reading shows that VF is present and that the medium sensitivity setting is sufficient at this stage.

The VF is still present at 121 but has developed into a lower amplitude or fine VF at 125. When this amplitude reduction occurs, the signal is "sub threshold" relative to the medium sensitivity signal. As a result, the TDO switches from high to low. At the reconfirmation point 121, the presence of the low TDO signal triggers a switchover from medium sensitivity level to high sensitivity level. The high sensitivity level picks up the lower amplitude fine VF and a high TDO reading is again generated.

Defibrillation shock therapy is given at 122. Low sensitivity subroutine IOI is accessed immediately post shock. The TDO signal goes low post shock showing the absence of VF, and sinus rhythm is detected at 123. Subroutine IOI then maintains, for the programmed timeout interval at 105, low sensitivity to distinguish R-waves from high amplitude current of injury T-waves.

If the medium sensitivity level were selected at 123, then double sensing of the R and T-waves would occur causing an incorrect reading along with correspondingly incorrect therapy which would cause severe problems and great discomfort to a patient. At 124 when the timeout for low sensitivity has elapsed (FIG. 5, at 105) there is a switchover from low sensitivity to medium sensitivity to pick up the R-waves of the normal sinus rhythm complex. If the duration of the timeout has been properly programmed, by this time the high amplitude current of injury T-wave has gradually decreased in amplitude to a normal amplitude T-wave.

The sensitivity levels in the device which are not variable but are held at fixed, discrete levels during normal operation are programmable by a physician. During programming or patient evaluation, an external programmer (not shown), which communicates with telemetry circuit 38, may be used for switching interchangeably from any one sensitivity level to any other sensitivity level.

Referring to Fig. 9A, there is depicted an ECG trace outlining a bradycardia pacing sequence after a defibrillation shock with a post shock pacing delay. At 130, a VT/VF arrhythmia has developed. Defibrillation shock therapy is applied at 131. As shown, the defibrillation shock, which has succeeded in reverting the VT/VF arrhythmia, is followed by a post shock pacing delay interval extending between 131 and 132. At 133, asystole is detected and bradycardia pacing is commenced approximately 4 seconds after the delivery of the defibrillation shock and continues at 134. The pro-arrhythmic effect of a premature recommencement of bradycardia support pacing immediately post reversion is avoided, as there is sufficient time for the conduction system of the patient's heart to be reorganized and susceptible to bradycardia support pacing.

Referring to Fig. 9B, there is depicted an ECG trace outlining a bradycardia pacing sequence after antitachycardia pacing with a post therapy pacing delay. At 140, a VT/VF arrhythmia has developed. Antitachycardia pacing therapy is applied at 141. As shown, the antitachycardia pacing, which has succeeded in reverting the VT/VF arrhythmia, is followed by a post therapy pacing delay extending from 141 to 142. At 143, asystole is detected and bradycardia pacing is commenced approximately 3 seconds after the termination of antitachycardia pacing and continues at 144. The pro-arrhythmic effect of a premature recommencement of bradycardia support pacing immediately after reversion has been avoided, as there has been sufficient time for the heart conduction system of the patient to be reorganized and susceptible to bradycardia support pacing.

In the description set forth above, the flow charts of FIG. 4, FIG. 5 and FIG. 6 implement sensitivity adjustment, and post therapy pacing delay. It will be recognized that either concept can be used independently of the other; that is an arrythmia control system may have multiple sensitivities without a post therapy delay, although a post therapy delay is preferable. Further, post therapy delay may be advantageously used in systems not having multiple sensitivities. For example, post therapy delay may be implemented in the system disclosed in the above-mentioned European Patent Application Serial No. 89300232.9 of Norma Louise Gilli, filed January 11, 1989 and entitled "Apparatus and Method for Controlling Pulse Energy in Antitachyarrythmia and Bradycardia Pacing Device."

As noted above, cardiac lead 12 may supply therapy to either the atrium or the ventricle. Thus the apparatus and method of the present invention is suitable for providing antitachyarrhythmia therapy to either the atrium or the ventricle or both.

Although the invention has been described with reference to a particular embodiment, it is to be understood that this embodiment is merely illustrative.

Claims

1. An apparatus (11) for reversion of tachyarrhythmia comprising:

   bradycardia support pacing means (35, 92) for providing bradycardia support pacing;
   detection means (37, 93) for detecting the presence of a tachyarrhythmia;
   therapy means (17, 44, 96) for delivering antitachyarrhythmia therapy;
   confirmation means (95, 97, 99) for providing confirmation of the reversion of said tachyrhythmia following delivery of said antitachyarrhythmia therapy;
   delay means (109, 4) for delaying, during a programmed, un-interruptible delay period substantially longer than one normal bradycardia support pacing interval, any determination of a need for therapy and any providing of therapy after said confirmation means has provided said confirmation of the reversion of said tachyarrhythmia; and
   means for automatically switching said apparatus into a bradycardia support pacing mode at the end of said delay period and for delivering any needed bradycardia support pacing using substantially a normal bradycardia support pacing standby interval, said automatic switching of said apparatus into said bradycardia support pacing mode occurring without further determination of whether or not there is a need for further antitachycardia therapy.

2. An apparatus as claimed in claim 1, wherein said delay means delays providing bradycardia support pacing for a delay period of between two and five seconds.

3. An apparatus as claimed in claim 1, wherein said antitachyarrhyhimia therapy means comprises at least one of an antitachycardia pacing pulse generation means (35, 36) and cardioversion shock means (17, 20) for delivering at least one cardioversion shock.

4. An apparatus as claimed in claim 3, wherein said delay means includes a first delay interval generation means for providing a first delay after antitachycardia pacing by said antitachycardia pacing pulse generation means, and a second delay interval generation means for providing a second delay

after delivery of said at least one cardioversion shock by said cardioversion shock means.

5. An apparatus as claimed in claim 4, further comprising means for independently setting said first delay interval generation means and said second delay interval generation means to provide said first delay interval of different duration than said second delay interval.

6. An apparatus as claimed in claim 4, wherein said first delay interval is of a duration of substantially three seconds.

7. An apparatus as claimed in claim 4, wherein said second delay interval is of a duration of substantially four seconds.

8. An apparatus as claimed in claim 1, wherein said antitachyarrhythmia therapy means delivers a first type of antitachyarrhythmia therapy and a second type of antitachyarrhythmia therapy, said delay means having a first delay interval generation means for delay after said first type of therapy and a second delay interval generation means for delay after said second type of therapy.

9. An apparatus as claimed in claim 8, further comprising programming means for independently programming the delay of said first delay interval generation means and the delay of said second delay interval generation means.

10. An apparatus as claimed in claim 1, further comprising programming means for programming the duration of said delay period.

Patentansprüche

1. Vorrichtung (11) zur Beseitigung von Tachyarrhythmie, welche aufweist:

   eine Bradykardie-Unterstützungsreizungseinrichtung (35, 92) zum Liefern einer Bradykardie-Unterstützungsreizung;
   eine Erfassungseinrichtung (37, 93) zum Erfassen des Vorliegens einer Tachyarrhythmie;
   eine Therapieeinrichtung (17, 44, 96) zum Liefern einer Antitachyarrhythmie-Therapie;
   eine Bestätigungseinrichtung (95, 97, 99) zum Liefern einer Bestätigung der Beseitigung der Tachyarrhythmie nach der Lieferung der Antitachyarrhythmie-Therapie;
   eine Verzögerungseinrichtung (109, 4) zum Verzögern während einer programmierten, ununterbrechbaren Verzögerungsperiode, die im wesentlichen länger als ein normales Bradykardie-Unterstützungsreizungsintervall ist, jeglicher Bestimmung einer Notwendigkeit einer Therapie und jeglicher Bereitstellung der Therapie, nachdem die Bestätigungseinrichtung die Bestätigung der Beseitigung der Tachyarrhythmie geliefert hat; und
   eine Einrichtung zum automatischen Umschalten der Vorrichtung in einen Bradykardie-Unterstützungsreizungsmodus am Ende der Verzögerungsperiode und zum Liefern jeglicher benötigten Bradykardie-Unterstützungsreizung unter Verwendung von im wesentlichen einem normalen Bradykardie-Unterstützungsreizungs-Standbyintervall, wobei das automatische Umschalten der Vorrichtung in den Bradykardie-Unterstützungsreizungsmodus ohne weitere Bestimmung, ob es eine Notwendigkeit für eine weitere Antitachykardie-Therapie gibt oder nicht, auftritt.

2. Vorrichtung nach Anspruch 1, wobei die Verzögerungseinrichtung eine Bradykardie-Unterstützungsreizung für eine Verzögerungsperiode im Bereich von zwei und fünf Sekunden liefert.

3. Vorrichtung nach Anspruch 1, wobei die Antitachyarrhythmie-Therapieeinrichtung zumindest eine der folgenden Einrichtungen aufweist: eine Antitachykardie-Reizungsimpulserzeugungseinrichtung (35, 36) und eine Kardioversionsschockeinrichtung (17, 20) zum Liefern von zumindest einem Kardioversionsschock.

4. Vorrichtung nach Anspruch 3, wobei die Verzögerungseinrichtung eine Erzeugungseinrichtung für ein erstes Verzögerungsintervall zum Liefern einer ersten Verzögerung nach der Antitachykardiereizung durch die Antitachykardie-Reizungsimpulserzeugungseinrichtung sowie eine Erzeugungseinrichtung für ein zweites Verzögerungsintervall zum Liefern einer zweiten Verzögerung nach der Lieferung des zumindest einen Kardioversionsschocks durch die Kardioversionsschockeinrichtung aufweist.

5. Vorrichtung nach Anspruch 4, welche weiterhin eine Einrichtung zum unabhängigen Einstellen der Erzeugungseinrichtung für das erste Verzögerungsintervall und der Erzeugungseinrichtung für das zweite Verzögerungsintervall zum Liefern des ersten Verzögerungsintervalls mit einer unterschiedlichen Dauer im Vergleich zum zweiten Verzögerungsintervall aufweist.

6. Vorrichtung nach Anspruch 4, wobei das erste Verzögerungsintervall eine Dauer von im wesentlichen drei Sekunden aufweist.

7. Vorrichtung nach Anspruch 4, wobei das zweite

Verzögerungsintervall eine Dauer von im wesentlichen vier Sekunden aufweist.

8. Vorrichtung nach Anspruch 1, wobei die Antitachyarrhythmie-Therapieeinrichtung einen ersten Typ von Antitachyarrhythmie-Therapie und einen zweiten Typ von Antitachyarrhythmie-Therapie liefert, wobei die Verzögerungseinrichtung eine Erzeugungseinrichtung für ein erstes Verzögerungsintervall für eine Verzögerung nach dem ersten Typ von Therapie und eine Erzeugungseinrichtung für ein zweites Verzögerungsintervall für eine Verzögerung nach dem zweiten Typ von Therapie aufweist.

9. Vorrichtung nach Anspruch 8, welche weiterhin eine Programmiereinrichtung zum unabhängigen Programmieren der Verzögerung der Erzeugungseinrichtung für das erste Verzögerungsintervall und der Verzögerung der Erzeugungseinrichtung für das zweite Verzögerungsintervall aufweist.

10. Vorrichtung nach Anspruch 1, welche weiterhin eine Programmiereinrichtung zum Programmieren der Dauer der Verzögerungsperiode aufweist.

Revendications

1. Appareil (11) pour une réversion de tachyarythmie, comprenant:

des moyens de stimulation de support de bradycardie (35, 92) pour assurer une stimulation de support de bradycardie;
des moyens de détection (37, 93) pour détecter la présence d'une tachyarythmie;
des moyens de thérapie (17, 44, 96) pour délivrer une thérapie antitachyarythmie;
des moyens de confirmation (95, 97, 99) pour produire une confirmation de la réversion de ladite tachyarythmie suite à la délivrance de ladite thérapie antitachyarythmie;
des moyens de retard (109, 4) pour retarder, pendant une période de retard non interruptible programmée sensiblement plus longue qu'un intervalle de stimulation de support de bradycardie normale, une quelconque détermination de la nécessité d'une thérapie et une quelconque administration d'une thérapie après que lesdits moyens de confirmation ont produit ladite confirmation de la réversion de ladite tachyarythmie; et
des moyens pour commuter automatiquement ledit appareil dans un mode stimulation de support de bradycardie à la fin de ladite période de retard et pour délivrer une quelconque stimulation de support de bradycardie nécessaire en utilisant sensiblement un intervalle d'attente de stimulation de support de bradycardie normale, ladite commutation automatique dudit appareil dans ledit mode de stimulation de support de bradycardie se produisant sans détermination supplémentaire de si oui ou non il y a la nécessité d'une thérapie antitachycardie supplémentaire.

2. Appareil selon la revendication 1, dans lequel lesdits moyens de retard retardent l'application d'une stimulation de support de bradycardie d'une période de retard comprise entre 2 et 5 secondes.

3. Appareil selon la revendication 1, dans lequel lesdits moyens de thérapie antitachyarythmie comprennent au moins des moyens pris parmi des moyens de génération d'impulsion de stimulation antitachycardie (35, 36) et des moyens de choc de cardioversion (17, 20) pour délivrer au moins un choc de cardioversion.

4. Appareil selon la revendication 3, dans lequel lesdits moyens de retard incluent des moyens de génération d'intervalle de premier retard pour produire un premier retard après une stimulation antitachycardie par lesdits moyens de génération d'impulsion de stimulation antitachycardie et des moyens de génération d'intervalle de second retard pour produire un second retard après la délivrance dudit au moins un choc de cardioversion par lesdits moyens de choc de cardioversion.

5. Appareil selon la revendication 4, comprenant en outre des moyens pour établir indépendamment lesdits moyens de génération d'intervalle de premier retard et lesdits moyens de génération d'intervalle de second retard pour produire ledit intervalle de premier retard de telle sorte qu'il soit d'une durée différente de celle dudit intervalle de second retard.

6. Appareil selon la revendication 4, dans lequel ledit intervalle de premier retard est d'une durée de sensiblement 3 secondes.

7. Appareil selon la revendication 4, dans lequel ledit intervalle de second retard est d'une durée de sensiblement 4 secondes.

8. Appareil selon la revendication 1, dans lequel lesdits moyens de thérapie antitachyarythmie délivrent un premier type de thérapie antitachyarythmie et un second type de thérapie antitachyarythmie, lesdits moyens de retard comportant des moyens de génération d'intervalle de premier retard pour un retard après ledit premier type de thérapie et des moyens de génération d'intervalle de second retard pour un retard après ledit second type de thérapie.

9. Appareil selon la revendication 8, comprenant en outre des moyens de programmation pour programmer indépendamment le retard desdits moyens de génération d'intervalle de premier retard et le retard desdits moyens de génération d'intervalle de second retard.

10. Appareil selon la revendication 1, comprenant en outre des moyens de programmation pour programmer la durée de ladite période de retard.

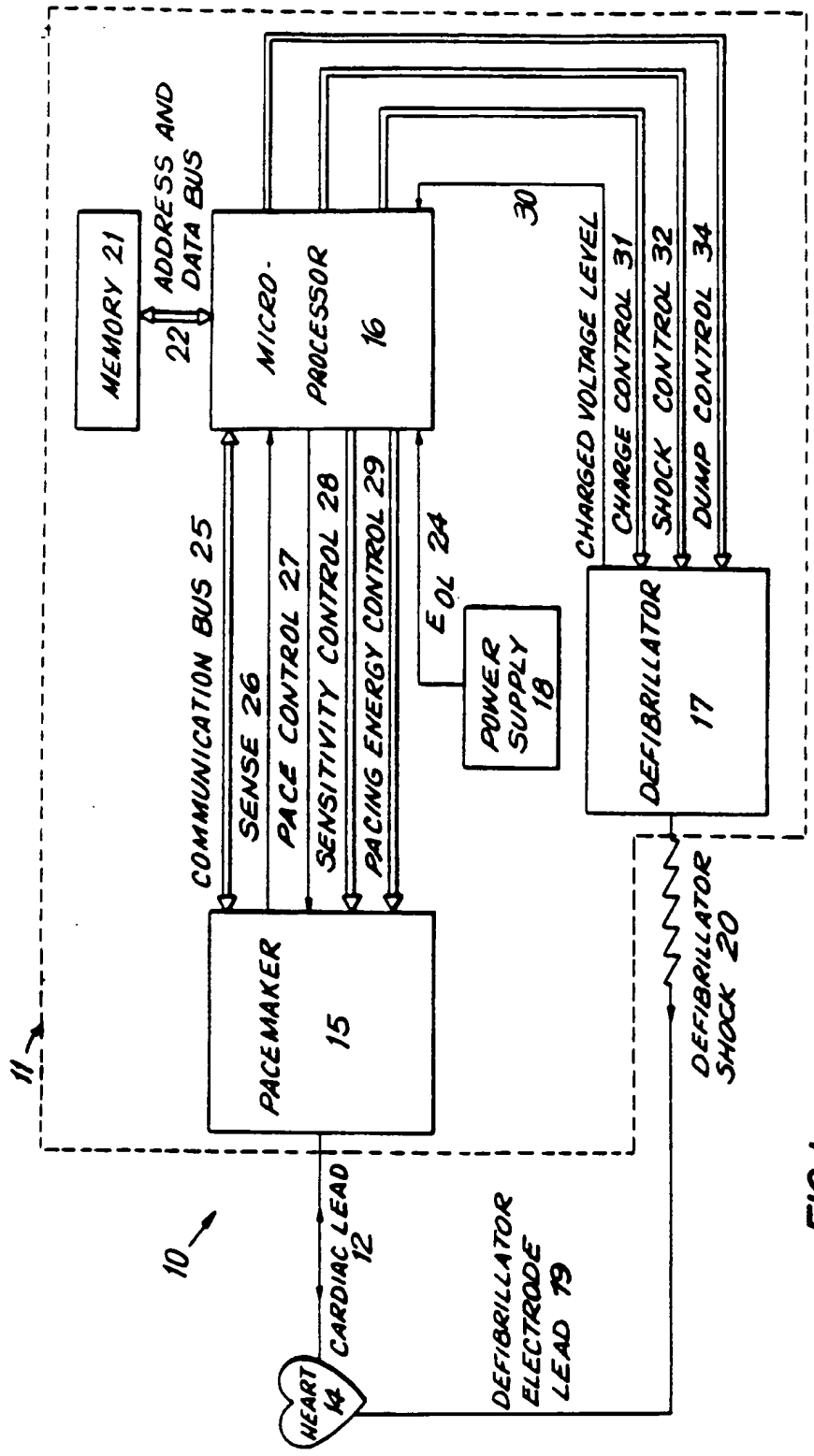
11
10
MEMORY 21
22
ADDRESS AND DATA BUS
MICRO-PROCESSOR 16
30
CHARGED VOLTAGE LEVEL
CHARGE CONTROL 31
SHOCK CONTROL 32
DUMP CONTROL 34
COMMUNICATION BUS 25
SENSE 26
PACE CONTROL 27
SENSITIVITY CONTROL 28
PACING ENERGY CONTROL 29
EOL 24
POWER SUPPLY 18
DEFIBRILLATOR 17
PACEMAKER 15
DEFIBRILLATOR SHOCK 20
CARDIAC LEAD 12
DEFIBRILLATOR ELECTRODE LEAD 19
HEART 14

FIG. 1

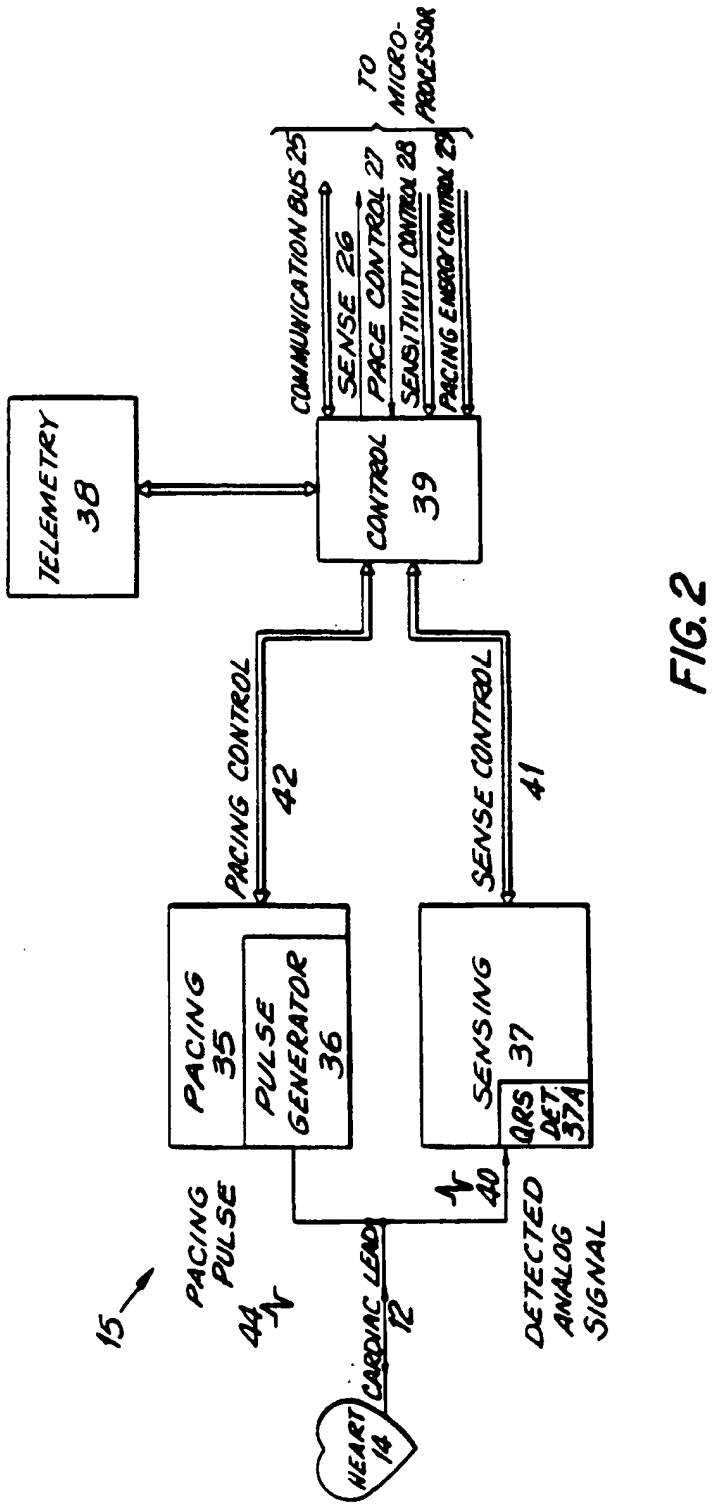
TELEMETRY
38
CONTROL
39
COMMUNICATION BUS 25
SENSE 26
PACE CONTROL 27
SENSITIVITY CONTROL 28
PACING ENERGY CONTROL 29
TO MICRO-PROCESSOR
PACING CONTROL
42
SENSE CONTROL
41
PACING
35
PULSE GENERATOR
36
SENSING
37
QRS DET 37A
15
PACING PULSE
44
CARDIAC LEAD
12
HEART
14
40
DETECTED ANALOG SIGNAL

FIG. 2

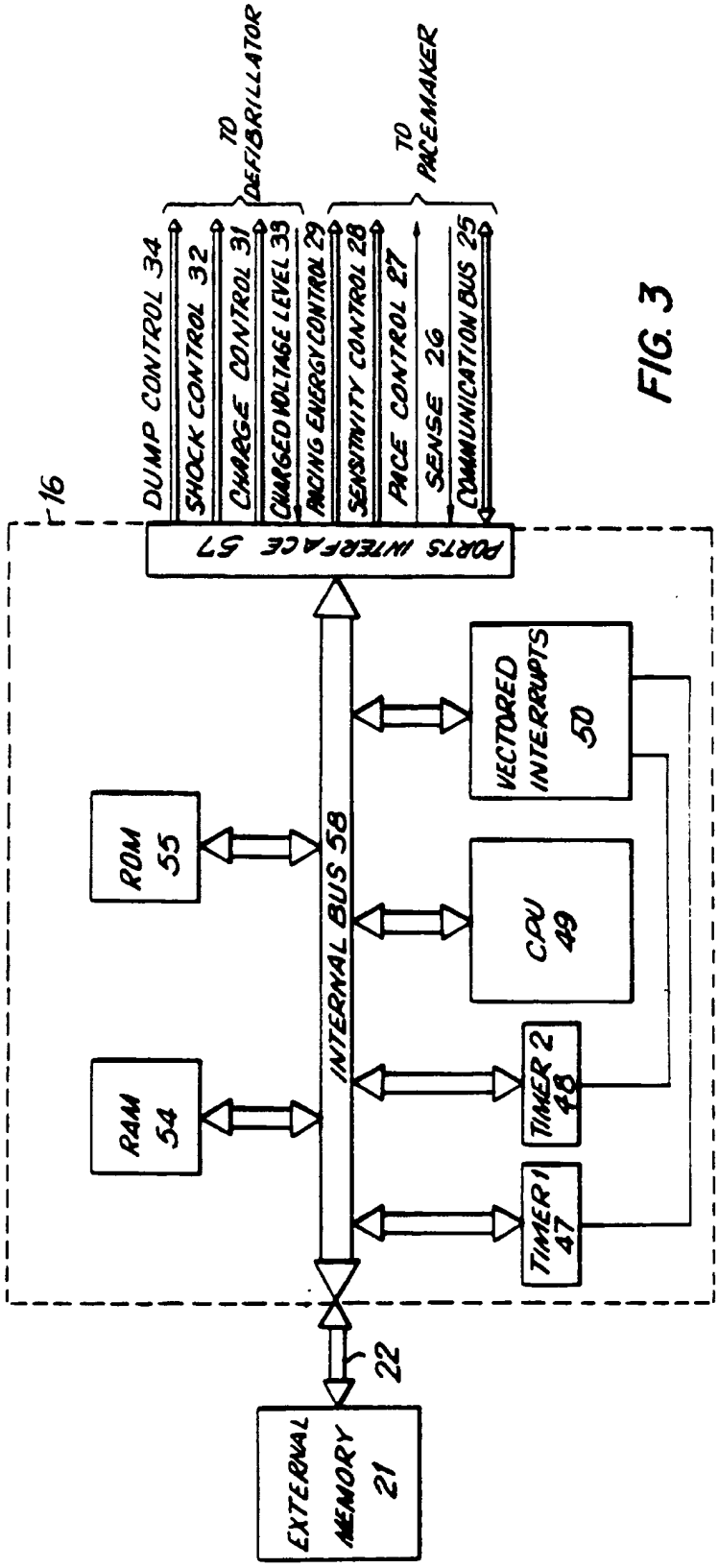
TO DEFIBRILLATOR
TO PACEMAKER
DUMP CONTROL 34
SHOCK CONTROL 32
CHARGE CONTROL 31
CHARGED VOLTAGE LEVEL 33
PACING ENERGY CONTROL 29
SENSITIVITY CONTROL 28
PACE CONTROL 27
SENSE 26
COMMUNICATION BUS 25
16
PORTS INTERFACE 57
VECTORED INTERRUPTS 50
ROM 55
CPU 49
INTERNAL BUS 58
RAM 54
TIMER 2 48
TIMER 1 47
22
EXTERNAL MEMORY 21

FIG. 3

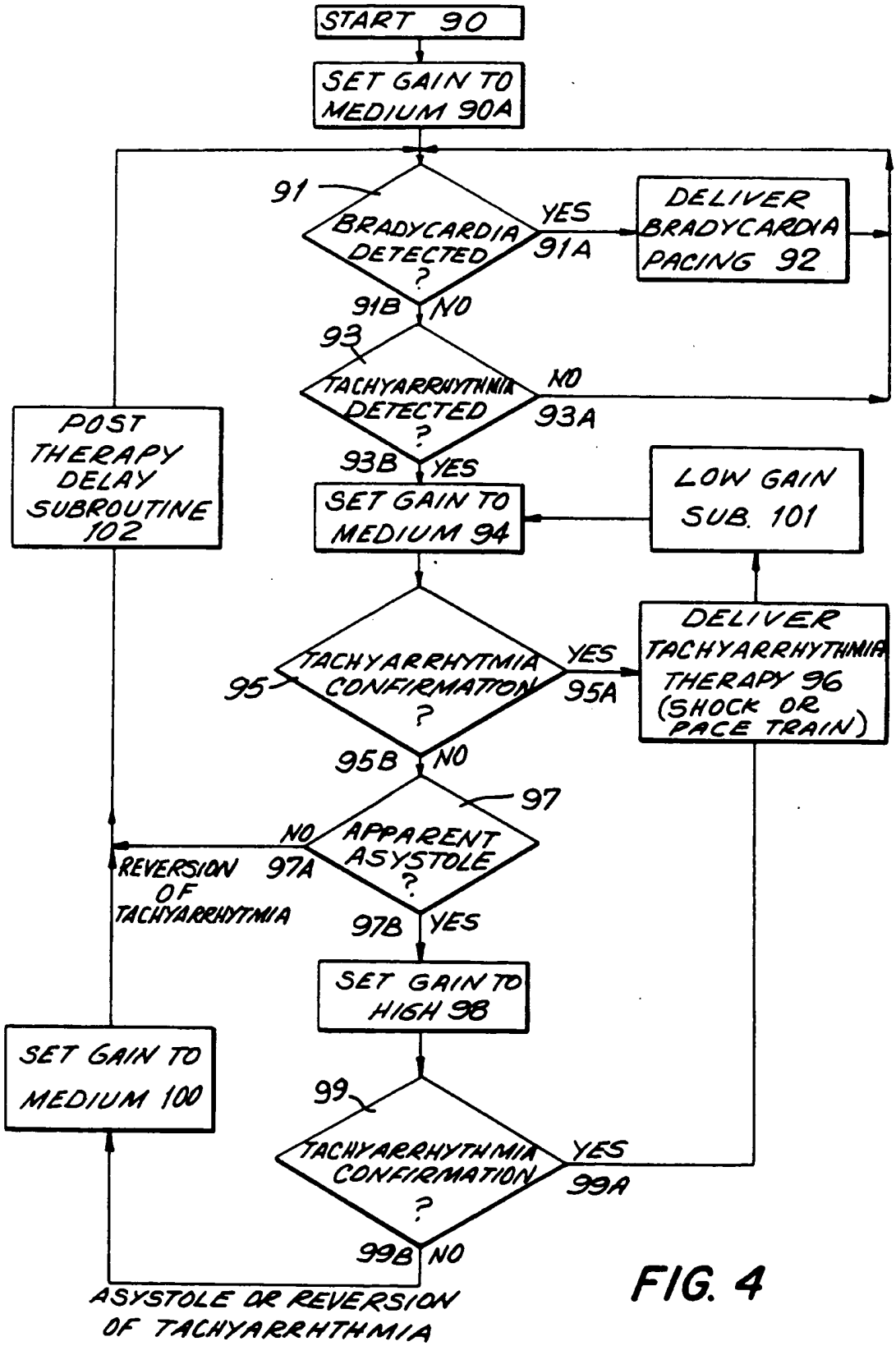
START 90
SET GAIN TO MEDIUM 90A
91
BRADYCARDIA DETECTED ?
YES
91A
DELIVER BRADYCARDIA PACING 92
91B
NO
93
TACHYARRHYTHMIA DETECTED ?
NO
93A
93B
YES
POST THERAPY DELAY SUBROUTINE 102
SET GAIN TO MEDIUM 94
LOW GAIN SUB. 101
TACHYARRHYTMIA CONFIRMATION ?
95
YES
95A
DELIVER TACHYARRHYTHMIA THERAPY 96 (SHOCK OR PACE TRAIN)
95B
NO
97
APPARENT ASYSTOLE ?
NO
97A
REVERSION OF TACHYARRHYTMIA
97B
YES
SET GAIN TO HIGH 98
SET GAIN TO MEDIUM 100
99
TACHYARRHYTHMIA CONFIRMATION ?
YES
99A
99B
NO
ASYSTOLE OR REVERSION OF TACHYARRHTHMIA

FIG. 4

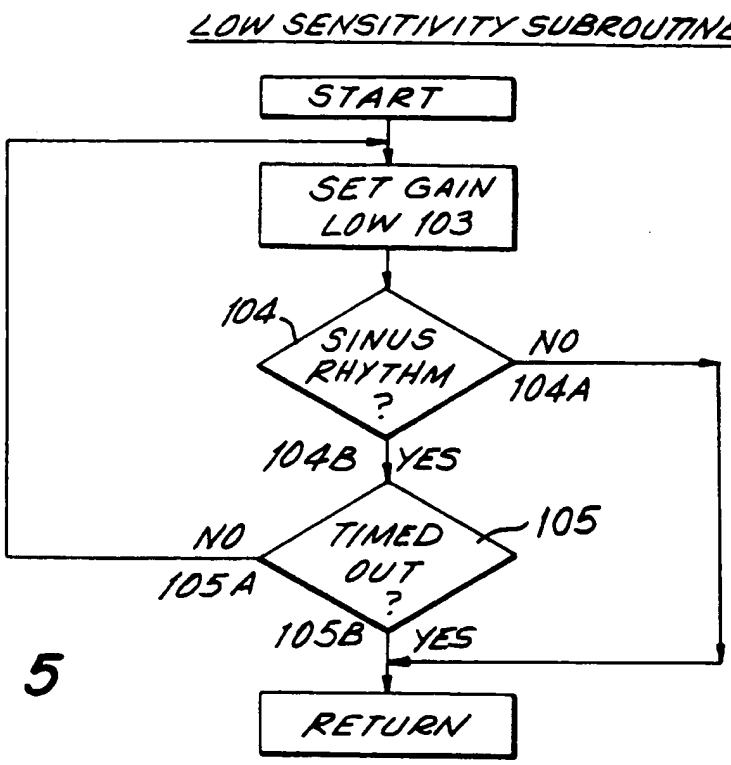
LOW SENSITIVITY SUBROUTINE
START
SET GAIN LOW 103
104
SINUS RHYTHM ?
NO
104A
104B
YES
TIMED OUT ?
105
NO
105A
105B
YES
RETURN

FIG. 5

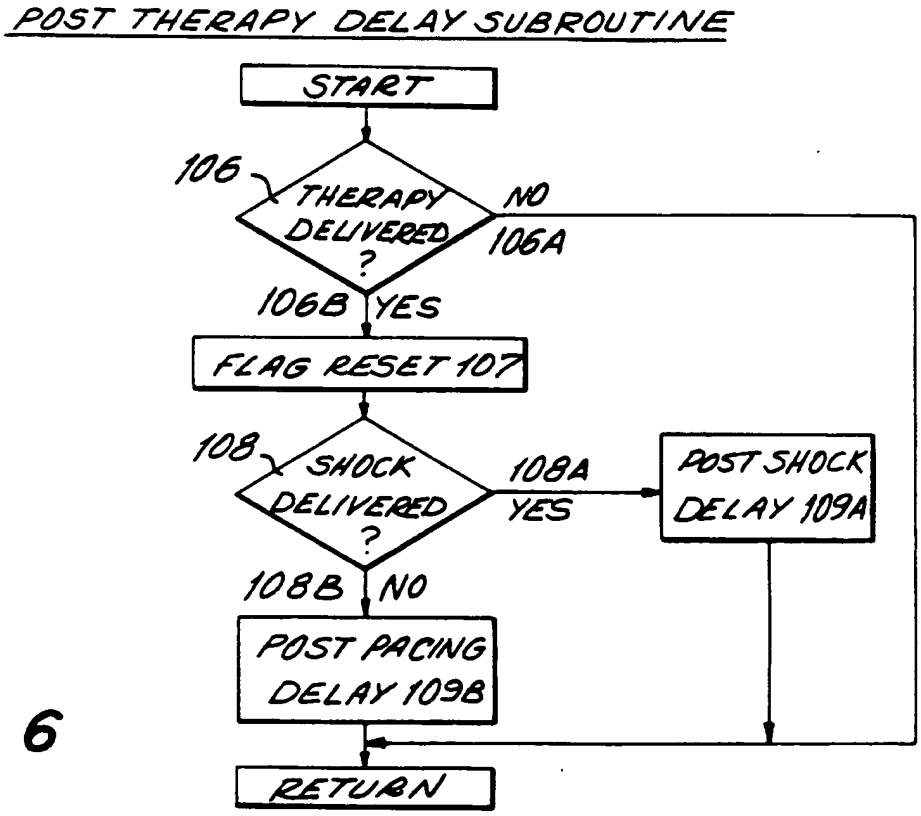
POST THERAPY DELAY SUBROUTINE
START
106
THERAPY DELIVERED ?
NO
106A
106B
YES
FLAG RESET 107
108
SHOCK DELIVERED ?
108A
YES
POST SHOCK DELAY 109A
108B
NO
POST PACING DELAY 109B
RETURN

FIG. 6

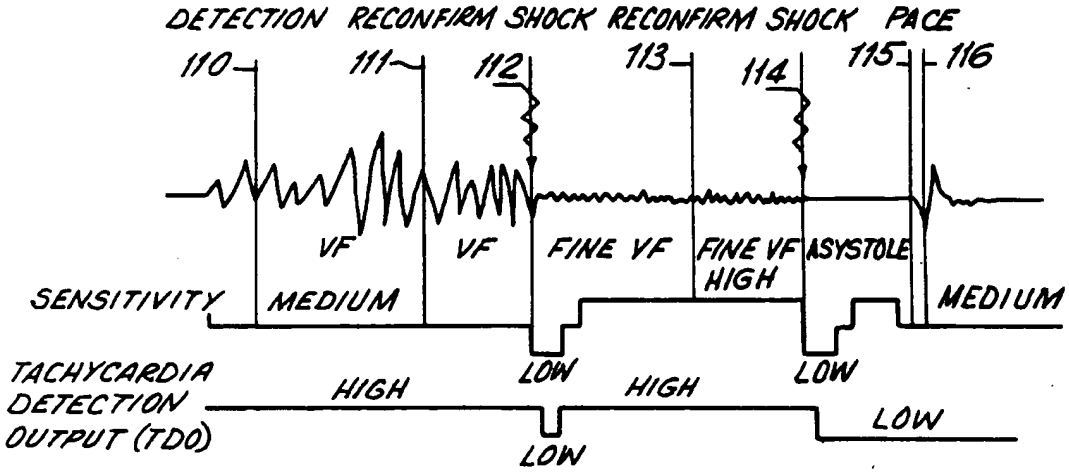
DETECTION
RECONFIRM SHOCK
RECONFIRM SHOCK
PACE
110
111
112
113
114
115
116
VF
VF
FINE VF
FINE VF
HIGH
ASYSTOLE
SENSITIVITY
MEDIUM
MEDIUM
LOW
LOW
TACHYCARDIA
DETECTION
OUTPUT (TDO)
HIGH
HIGH
LOW
LOW

FIG. 7

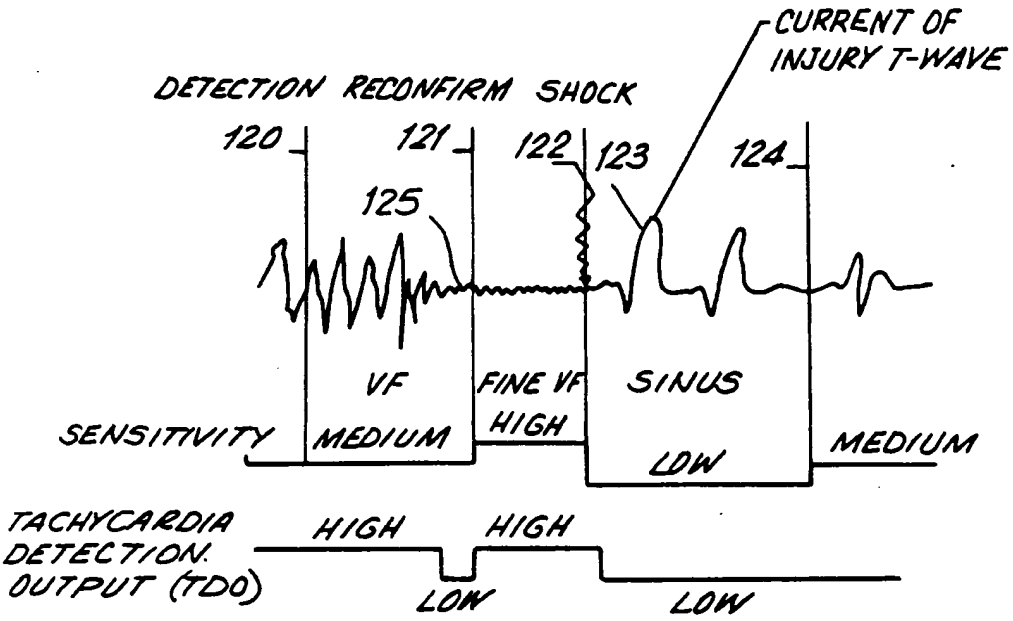
CURRENT OF
INJURY T-WAVE
DETECTION
RECONFIRM
SHOCK
120
121
122
123
124
125
VF
FINE VF
SINUS
SENSITIVITY
MEDIUM
HIGH
LOW
MEDIUM
TACHYCARDIA
DETECTION.
OUTPUT (TDO)
HIGH
HIGH
LOW
LOW

FIG. 8

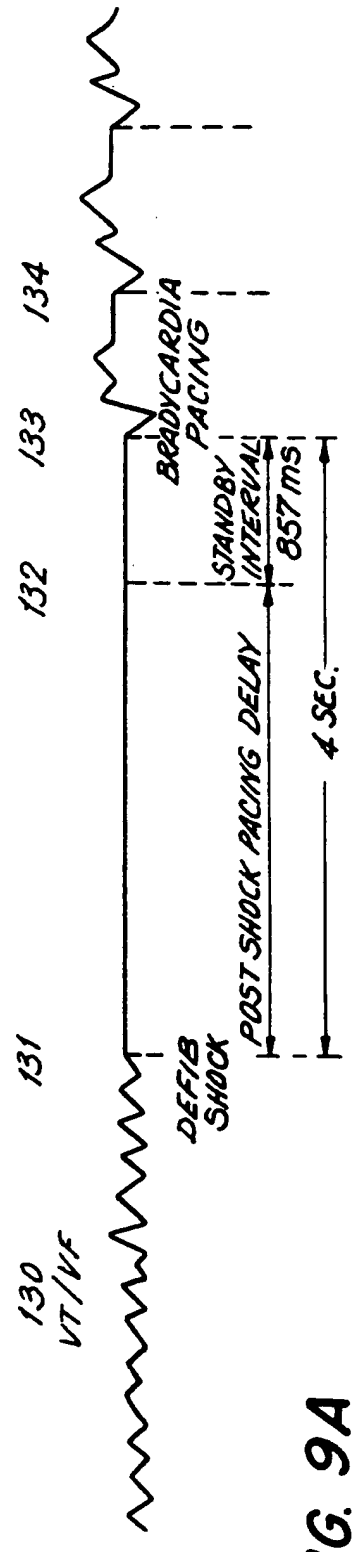
130
VT/VF
131
DEFIB SHOCK
132
POST SHOCK PACING DELAY
133
STANDBY INTERVAL
857 ms
134
BRADYCARDIA PACING
4 SEC.

FIG. 9A

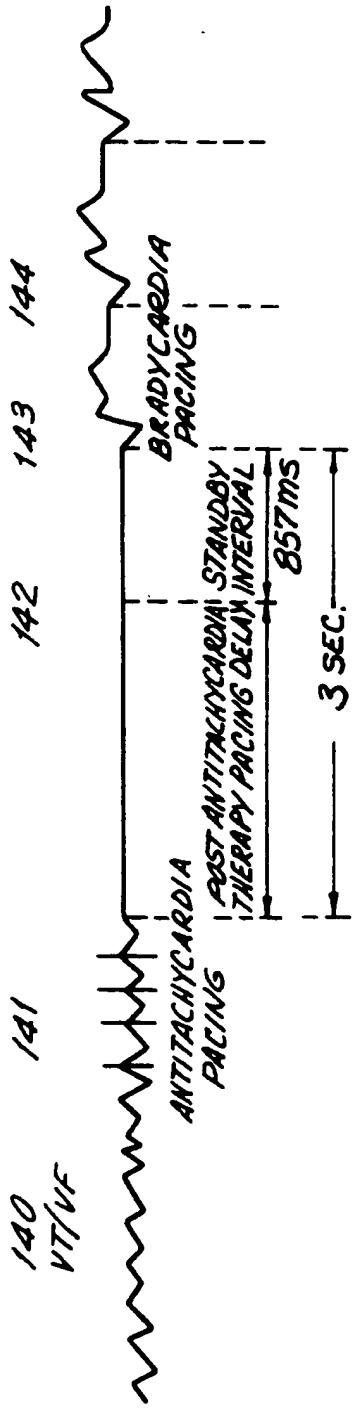
140
VT/VF
141
ANTITACHYCARDIA PACING
142
POST ANTITACHYCARDIA THERAPY PACING DELAY
143
STANDBY INTERVAL
857ms
144
BRADYCARDIA PACING
3 SEC.

FIG. 9B